ACQUIRED DIVERTICULA OF THE SIGMOID, WITH A REPORT OF SIX CASES.[1]

By Arthur D. Dunn, M.D.,
PROFESSOR OF MEDICINE, JOHN A. CREIGHTON MEDICAL COLLEGE, OMAHA, NEBRASKA.

AND

Paul G. Woolley, M.D.,
PROFESSOR OF PATHOLOGY, OHIO-MIAMI COLLEGE OF MEDICINE, UNIVERSITY OF CINCINNATI;
DIRECTOR OF THE LABORATORIES OF THE CINCINNATI HOSPITAL.

At the Twenty-seventh Congress of Surgery, Graser demonstrated a stenosis of the sigmoid due to chronic hyperplastic inflammation, on which an operation for cancer had been performed. He believed the portal of entrance for the infection to be a number of acquired diverticula, some of which were seen entering the inflammatory mass. From Graser's paper dates the clinical interest in the subject, for before that time diverticula had been considered merely pathological curiosities. They had been described by Morgagni (1796), Fleischmann (1815), Albers (1844), Habershon (1857), Klebs (1869), Loomis (1872), Fischer (1900), and others.

Acquired diverticula are of two varieties, the true and the false. True diverticula are merely wide-mouthed pouches whose walls contain all of the layers of the normal bowel. They are the result of an abnormal tendency to haustrum formation, and seem to be of anatomical significance only. False diverticula, on the contrary, are herniaform protrusions of the mucous membrane through the muscularis, and are of increasing clinical and pathological moment.

Acquired diverticula may occur throughout the entire intestinal tract. They have frequently been seen in the duodenum in the vicinity of Vater's papilla. They are common in the colon, and they cease abruptly at the first portion of the rectum. Occasionally they have been observed in the appendix, where they may be the seat of a condition which cannot be differentiated from appendicitis. They vary in number from several hundred (400, Hansemann) to an isolated protrusion. They are generally multiple.

Since diverticula of the sigmoid are much more likely to give rise to clinical manifestations, your attention will be directed toward them only.

Etiology. Their origin is still a mooted question. A predisposition to diverticulum formation undoubtedly exists, and according to Graser and Sudsuki is common. On microscopic examination of the sigmoid in old subjects, the former found small diverticula in 10 out of 28 cases, and the latter in 15 out of 40 cases.

---

[1] Read before the Cincinnati Society for Medical Research, February 24, 1910.

The following theories as to cause have been suggested:

1. That the fenestra through which the vessels penetrate the muscularis form *loci minoris resistentiæ*. The vessels are surrounded by loose connective tissue which contains a variable amount of fat. As the individual ages, the fat increases, the connective tissue becomes weaker, and the fenestra are increased in size by atrophy of the muscularis. Intermittent stasis also stretches and increases the diameter of the fenestra. During periods of cessation of stasis, the caliber of the vessels diminish, and loose space in the "vessel holes" results. These factors increase the predisposition. Accumulation of feces and gas incident to constipation, bowel atony, and abnormal decomposition act to a degree as determinining factors. The vascular theory is accepted by Fischer, Koch, Hansemann, Berner, and others, and is corroborated by four of the cases which we studied microscopically.

2. Bland-Sutton believes that the appendices epiploicæ form points of lessened resistance because, as he has shown, their fat is in immediate continuity with the subserous fat of the intestinal wall. It is difficult to see, however, why continuity of fat should predispose to diverticula. The process is that of a penetration of the muscularis, and it seems highly improbable that the weak serosa, and its subserous layer of fat, could play any part in it. It is more probable that the fact that bloodvessels penetrate the muscularis at this point is the chief reason for the common incidence of diverticula here. In some cases an adherent appendix may cause the production of traction diverticula.

3. Franke believes that developmental anomalies are at the basis of diverticulum formation. What these developmental imperfections are he leaves unspecified.

4. Beer and Telling consider weakening of the muscular coat in adult life as the chief cause of diverticula. They do not state why the muscular wall should weaken in certain spots and not in others. They assume that the vessels may "direct the path" for the diverticula. Beer states: "The fact that diverticula occur in old people, in people whose intestines have been more or less worked out, as evidenced by constipation, points to a muscular deficiency, and in this muscular weakness the cause of false diverticula must be sought."

5. Age seems to be to some extent a determining factor. In 80 cases the average age was sixty years (Telling). Forty-seven of the 80 cases gave rise to clinical symptoms at the average of fifty-five years. The ages of our 6 cases were as follows: Without symptoms, 75, 78, and unknown; with symptoms, 83 (symptoms due to cicatricial stenosis secondary to diverticulitis), 45, and 50.

6. Sex also plays a part. Of 81 cases, 53 were male, 28 female (Telling). Our 6 cases were all men.

7. Obesity has been emphasized as a causative factor by Klebs, Mayo, and others, but it seems to be only an indication of involution

processes and thus only a concomitant of weakness in the muscularis or in the "vessel holes." Only 2 of our cases showed any unusual increase in fat.

8. Cachexia and absence of fat were marked in one of our cases. It might be inferred that in individuals who had been obese the loss of fat would tend to weaken the intestinal wall by leaving unoccupied or loose spaces.

9. The physiological role of the sigmoid must be of some moment in promoting diverticulum formation. The feces are retained longest in this segment (average ten to twenty-four hours), and its walls are incessantly subjected to changes in pressure due to variations in the quantity and quality of its contents, and so a predisposition and tendency to atony is inherent in the normal function of the sigmoid.

10. When the physiological retention of feces becomes excessive and constipation arises, the strain upon the gut walls is abnormal and any tendency to atony is aggravated. Localized weakenings are subjected to a determinative factor. The causal relationship of constipation to diverticulum formation so often observed might seem to support this view. Fischer's doubts as to the importance of constipation, however, are well founded. Its role has certainly been exaggerated and cases are numerous in which constipation has not been recorded. Only a very small proportion of the constipated have diverticula. Abnormal decomposition of retained contents with gas production is probably of more moment. Constipation, if it plays a part, must apparently be of the atonic type. The writers know of no investigation as to this point. Any factor, however, that will increase intrasigmoidal pressure must influence the development of diverticula at points of lessened resistance.

We may briefly summarize the etiological factors, all of which may be present to a variable extent in every case, as follows: (1) Congenital or acquired anatomical predisposition to localized weakness in the wall of the gut. (2) The addition of certain positive mechanical factors to the above potential factors, viz., (*a*) Strain on the bowel wall due to the accumulation of gas and feces. (*b*) Changes in the caliber and structural strength of the "vessel holes" consequent on stasis and senile circulatory disturbances, or on rapid loss of visceral fat. (*c*) Atony, which may be due to age, distention, or obesity.

PATHOLOGY. There is little definite information on the postmortem incidence of acquired diverticula of the sigmoid, for, as a rule, little attention is given to this segment of the gut in the routine autopsy, unless clinical manifestations or conspicuous pathological alteration draws the attention thither. In 78 cases in which the sigmoid was carefully examined by us at the autopsy, acquired diverticula were found five times. Two of the above cases were clinical. In one, the diagnosis was suspected almost to a certainty, in the second, it was a "find." The remaining cases were discovered because they were looked for.[2] False diverticula are usually situated near the mesenteric attachment. According to Bland-Sutton, Franke, and Telling, they are most commonly formed at the appendices epiploicæ. In three of our autopsies they were located at these points. In one case their location was at the mesenteric attachment. In one case, the diverticula were located in the vicinity of the appendices, but their relationship to them could not be determined on account of the hyperplasia of the gut wall and an inflammatory infiltration of the immense amount of fat surrounding the sigmoid. One peculiarity in this case not hitherto noted is the location of the diverticula on the summits of the rugæ, where they were discovered as minute depressions only after careful search. One of these innocent-looking depressions led into a fistulous tract. Location opposite the mesenteric attachment has been described by Hansemann.

The *size* of *acquired diverticula* varies from minute herniaform protrusions of the mucosa, discoverable only with the microscope, to processes the size of peas or grapes. (Size of an apple—Edel; of an egg—Virchow). They are conical, sacular, spherical, or teat-like processes

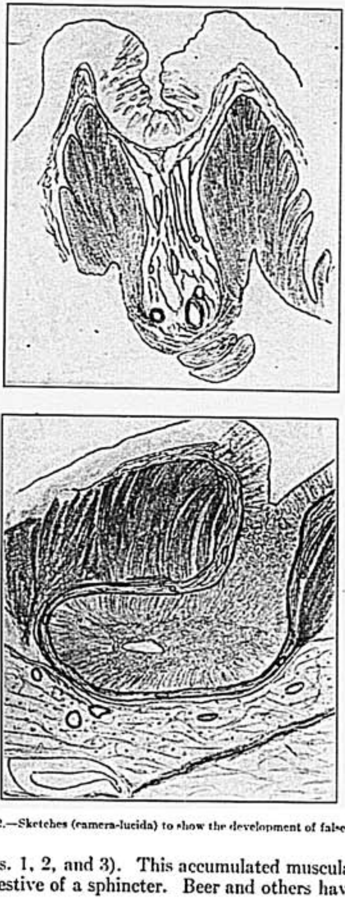
The ostia vary markedly in size. Sometimes the diameter of the opening equals that of the average diameter of the diverticulum. Often the openings are narrow, and although one easily discovers the pouch on the outer surface of the bowel, the mucosa must be scanned carefully to find the mouth. Many of them are distinctly flask-shaped, and their necks seem to be constricted by the penetrated musculature. This shape is especially characteristic of diverticula into the appendices epiploicæ, and creates an excellent mechanism to prevent drainage. Catarrhal inflammation may easily cause edema and swelling of the mucosa, and thus completely occlude the neck. Thus, there is formed a thin-walled sack filled with infectious material lying outside of the gut. The peritoneal cavity is protected only by a thin membrane consisting of mucosa, serosa, and possibly a few attenuated muscle fibers. The mucosa itself is often eroded or atrophic. It may be completely denuded. Localized foci of infection are common.

The coats of the diverticulum vary. Mucosa, submucosa, and serosa are constant. The amount of muscularis varies in different diverticula, and in different parts of the same diverticulum. The walls are thinnest at the tips of the processes.

Here there may be nothing but a thinned outer layer of mucosa and serosa. In our sections the penetration of the muscularis is clear, even though the diverticulum carries some muscle fiber with it. The contrast in the thickness of the muscular coating of the diverticulum, and of the layers through which it passes, is most

---

[2] Since the above was written one case has been found in the pathological service of Dr. Woolley in the Cincinnati Hospital, the first observation in 103 autopsies. No symptoms were referable to the diverticulum.



FIGS. 1 and 2.—Sketches (camera-lucida) to show the development of false diverticula.

marked (Figs. 1, 2, and 3). This accumulated musculature at the neck is suggestive of a sphincter. Beer and others have suggested that the origin of this picture is a localized weakening of the muscularis, but this still leaves an explanation to be explained. The potentiality for mischief which lies in these structures is evident from an inspection of the sections shown with the description of our cases.

The morbidity from false diverticula depends on the following factors:

1. Thinning of the coats.
2. Ulcerative and perforative action of retained concretions and feces.
3. Presence of pathogenic microörganisms.
4. Defective drainage due to: (*a*) Lack of an effective muscular coat. (*b*) Closure of the neck by edema of the mucosa secondary to colitis or stasis. A mild catarrhal colitis may occlude the openings, and thus result in the formation of a pocket of infectious material, from which the peritoneal cavity is poorly protected by a coat of variable thinness. (*c*) Strangulation of the opening by kinking, torsion, or muscular (sphincter) action.

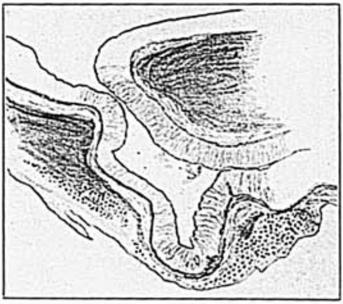


FIG. 3.—Sketch (camera-lucida) to show the development of a false diverticulum.

The following morbid processes, due to acquired diverticula of the sigmoid have been noted:

1. *Local Peritonitis.* Cases have been reported in considerable number. Abscesses in the left iliac fossa have frequently been drained and attributed to various causes, such as left-sided appendicitis, infected appendices epiploicæ, colonic ulceration, etc. This is probably the most frequently observed clinical manifestation of diverticulitis. (Telling collected 24 cases.) One of our cases was of this type.

2. *Acute General (Diffuse) Peritonitis.* (*a*) With perforation, 14 cases (Telling). (*b*) Without perforation. Loomis (1872) reports

a single case of general peritonitis in which diverticula were present in large numbers. He assumed that the infection traversed the thin wall of a diverticulum without rupture.

3. *Chronic Hyperplastic Sigmoiditis.* Within the last few years a number of cases have been reported, characterized by a productive inflammation of the sigmoid, which manifests itself by disturbed function of the lower bowel; pain, intermittent and colicky in character; tenderness; and constipation, often spastic in type and frequently alternating with diarrhea. Mucus and occasionally a trace of blood are present in the stools. Examination showed tenderness in the left iliac fossa with a movable sausage-shaped tumor. Recent observations prove a large percentage of these cases to be the result of diverticulitis and peridiverticulitis. Patel says: "Peute-être un jour sera-t-on droit de dire que dans presque tous ces cas decrites sous le nom de sigmoiditis, il s'agit de véritables divérticulites, absolument comme l'appendicite a été substituée peu à peu à la typhlite." There is little question but that many of the successful resections of the sigmoid for carcinoma have been cases of infiltrating sigmoiditis. A review of museum specimens (Mayo, Wilson, Telling, etc.), is already putting certain excised cancers of the sigmoid into this class. Such reports will increase in frequency as the condition becomes better known.

4. *Cancer.* Four cases are reported by Hochenegg, Stierlin, Telling, and Wilson,[2] in which cancer has developed on the basis of a sigmoiditis and perisigmoiditis secondary to chronic diverticulitis. The low grade of prolonged inflammatory irritation which so often precedes carcinoma elsewhere is here produced by a chronic diverticulitis.

5. *Stenoses.* These are the result of inflammation in the intestinal wall. Usually they are caused by a productive inflammation, *i. e.*, sigmoiditis and perisigmoiditis which narrows the lumen of the gut. The mucosa is thrown into folds, but, as a rule, is not ulcerated. Cicatricial contraction following a diverticulitis may produce stenosis, as is clearly shown by one of our specimens. Telling reports the only other case in the literature.

6. *Adhesions.* Adhesions produce symptoms by: (*a*) Traction and constriction. The obstruction is commonly due to the fact that the small bowel becomes adherent to the inflammatory area. (*b*) Adhesions of the gut to the bladder with vesicosigmoidal fistula. Telling found adhesions reported sixteen times and fistula eleven times.

7. *Chronic Mesenteritis.* Riedel, Brehms, Ries, Simpson, and Gardinier have called attention to the probable importance of sigmoid diverticulitis in the production of retraction and shortening

---

[2] Donald MacCrae, of Council Bluffs, in a personal communication, reports another case in which he excised a carcinoma of the sigmoid which was penetrated by numerous diverticula and which showed the carcinoma developing in inflammatory tissue.

of the sigmoid mesentery, a state which predisposes to volvulus. One of our cases was of this type.

8. *Metastatic Suppuration.* Whyte (1906) has reported the only case of this type. A limited necropsy disclosed multiple abscesses of the liver. The original focus was supposed to be an inflamed diverticulum of the sigmoid containing a fecal concretion.

9. *Perforation Into a Hernial Sac.* Stierlin has reported a case of suppuration into a hernial sac from a perforated diverticulum in an incarcerated sigmoid.

CASE I.—B. G., male, aged forty-five years. The family history is negative, and the personal history also until one and one-half years before the present illness, when the patient had an attack of pain and distress in the hypogastrium, with temperature. There was some diarrhea and tenesmus. Constipation was not present at any time. He went to bed for several days on a restricted diet and recovered rapidly.

Except a few fleeting pains and an occasional sense of fulness in the lower quadrants, which disappeared with catharsis, nothing of moment occurred for one and one-half years, when, after a few days of indefinite distress in the hypograstrium, he suffered severe pains in the lower abdomen, which were accompanied by dizziness, faintness, chill, temperature of 103°, and later sweating. The bowels had been free, but with some griping and soreness. The patient went to bed on a restricted diet, and for several days had an irregular temperature, ranging from 99° to 103.5°, accompanied by distress in the lower abdomen. Examination brought out the following facts:

The patient was a well-developed middle-aged man. His complexion was muddy, his tongue coated, and his breath foul. Examinations of the heart, nervous system, and lungs were negative except for the rapidity of the heart and for signs of retraction at the left apex. The abdomen was uniformly distended and tympanitic. There was diffuse tenderness below the umbilicus, which was best defined in the left iliac fossa, and in the median line, where he always referred his pain. No tumor mass could be demonstrated. A slight visceromotor reflex was present in both lower quadrants, but was most marked on the left. There was no area of cutaneous hyperesthesia. Neither testicle was tender. Rectal examination was negative. The leukocyte count was 15,000. The urine contained much indican, but was otherwise negative. The stools were very offensive, contained a little mucus, but neither blood nor pus. Large thick Gram-positive bacilli were present in large numbers. The pulse was rapid (100 to 130).

With colonic irrigation and on a diet of buttermilk and carbohydrates the patient slowly improved. For a month there were occasional attacks of pain, with a little temperature, which were relieved by flushings. Convalescence was slow. Several leukocyte counts during this period gave an average of 8000 to 10,000. Indican was more or less constantly present until convalescence was well established. A diagnosis of intestinal toxemia with possible sigmoiditis was made at that time.

The patient left home and did fairly well for one month, when he was taken with sudden, severe pain in the hypogastrium, hurried to the hospital, and was operated upon with the diagnosis of appendicitis. In a personal communication, the surgeon stated that "the appendix was thickened and slightly injected, but I must confess it hardly seemed sufficiently involved to account for the large amount of turbid fluid which we found in the lower abdomen. There were no adhesions about the cecum, which was apparently normal. I did not examine the sigmoid colon."

One month later the patient returned for treatment. His distress had returned after the operation, although not to the same degree. A strict dietetic and hygienic régime with colonic flushing gradually brought the patient into the best health he had enjoyed for several years. Strict injunctions against excessive indulgence in proteids, especially high meats and cheeses, were given, as experience had proved that a minimum of proteid was accompanied with less distress. With much proteid ingestion, indican always appeared in the urine and undigested meat fibers in the stools. When the diet was strictly followed, the stools lost their offensiveness, the gas diminished, and the number of Gram-positive bacilli decreased. Repeated rectal examinations were negative.

After three months of comparative freedom from trouble, the patient committed two dietetic indiscretions within twenty-four hours. Pain and distress in the hypogastrium followed, with temperature ranging from 99° to 104°, and a pulse of 100 to 120. His condition improved slowly for five days under treatment, when a sudden sharp pain followed an injection of water. The pain was so severe that morphine was given before the patient was seen. On examination there was considerable tenderness in the lower abdomen, some rigidity, but no cutaneous hyperesthesia. Rectal examination was negative. There was a little nausea, but no vomiting. A tumor mass could not be demonstrated at this time; there was some pain at the end of urination. On several occasions during the next few days the stools showed a little pus, mucus, and blood. Ten days later a mass had clearly defined itself in both iliac fossa, and could be detected per rectum as an irregular, elastic, bulging body. The patient's general appearance was decidedly septic, and he was removed to the hospital for operation. Leukocytes, 40,000.

From the clinical course it was assumed that the condition was a localized purulent peritonitis rising from the sigmoid. A chronic ulcerative process was excluded on account of the absence of blood and pus in the stools until the last few days. Perforation of a carcinoma was unlikely from the length of the clinical course absence of blood, the absence of a tumor until a few days previous, (two years), the absence of signs of stenosis, the almost entire the character of the tumor, and its rapid development. By exclusion, diverticulitis of the sigmoid with the perforation of an infected diverticulum resulting in a pelvic abscess was considered probable.

Operation. An incision was made in the median line, and inasmuch as the most prominent part of the tumor mass seemed to the surgeon to be on the right side, a wide incision was made well to the right of the rectus muscle, and free drainage instituted. The case passed from observation, and death took place about six weeks after the operation. The autopsy report was as follows:

Operation about ten hours after death. Skin very pale. Slight rigor mortis. Peripheral lymph glands not enlarged. Two operative wounds on abdomen—one in mid-line about 10 cm. long; one over appendix about 7.5 cm. long. The latter drained. Subcutaneous fat well developed.

Upon opening the peritoneal cavity it was seen that the omentum was adherent over the whole anterior surface of the peritoneum, and less extensively to the intestinal coils. It was rolled into a mass in the left hypochondrium.

The intestinal coils were more or less generally adherent by old fibrous adhesions, and were also adherent to the parietal peritoneum in the right flank. The coils of the ileum were closely adherent to one another, and the lower ones were also adherent to the sigmoid by old and recent adhesions.

The cecum was bound down by adhesions, most of them recent, the results of an operation done some days before to drain the cecal and pelvic regions. There was no pus in the pelvis or about the cecum. The appendix had been removed.

The intestines were carefully dissected out and no other process than an adhesive fibrosis was encountered until the lower loops of the small intestines were dissected away from the sigmoid, just at the brim of the pelvis. At this point an abscess cavity situated under the sigmoid and walled off by the small intestine was opened.

As the large intestine was removed, pus was again encountered to the left and beneath the descending colon and sigmoid. This suppurative process could be traced to the abscess cavity mentioned above and to a larger one to the left of it, in the postintestinal tissues and just at the brim of the pelvis. The process had apparently also extended down (judging from the adhesions), but this extension had been limited by adhesions in the pelvis. Extending up along and behind the descending colon the line of extension of suppuration could be followed to the spleen, which was half enclosed in an abscess cavity.

An incision through the diaphragm from below and exploration of the left pleural cavity occasioned a gush of foul-smelling, greenish-yellow, thin pus to the amount of about 500 c.c.

The thorax was then opened and examined. The left lung was partially compressed by an empyema that extended from the diaphragm to near the apex. The pleura was covered by a greenish-yellow fibrinopurulent exudate. The left apex was the seat of a large healed scar, and scattered about on the pleura and in the pulmonary tissue were fibroid and calcareous obsolescent tubercles.

The right lung was also the seat of a healed tuberculous process, the pleura being thickly studded with obsolescent tubercles, especially at the junction, posteriorly, of the interlobar clefts.

The heart was pale but otherwise normal.

The liver was small, pale, and exceedingly soft.

The kidneys were very large and pale. The capsules stripped, leaving an untorn surface. Section of the organ showed the cortex enlarged, pale, and granular in appearance, with little line of demarcation between it and the medulla.

The adrenals were normal.

The pancreas was normal.

The spleen was of normal size, but soft, and the seat of an acute suppurative perisplenitis.

When the intestines were opened it was seen that the small intestine was atrophic, the walls thinned, the rugæ not prominent, and the mucosa thinned. From the cecum down, the submucosa and the muscularis became gradually thickened until, in the vicinity of the brim of the pelvis, the wall was from two to five millimeters thick, and perisigmoidal and rectal tissues were generally firm and infiltrated.

The sigmoid and rectum showed accentuated diverticular pouches, all of which had thinned distal ends and mouths about which the submucosa and the muscularis were very thick and edematous (Fig. 4). Extending from the distal end of one of these pouches was a sinus, surrounded by thickened fibroid walls, that communicated with the larger postcolonic abscess cavity. From this pouch or diverticulum the suppurative process had apparently originated. The tops of the folds of the descending colon and sigmoid showed numbers of small areas having somewhat the appearance of shallow ulcers. These areas measured not more than one by two millimeters. They had slightly thickened, raised, and rounded margins, and gray bases.

Anatomical Diagnosis. Anemia; catarrhal sigmoiditis and proctitis; diverticulum formation; chronic perforative diverticulitis; suppurative perisigmoiditis, pericolitis and perisplenitis; empyema; parenchymatous degeneration of the kidneys; fatty degeneration of the liver; obsolescent, calcareous pulmonary tuberculosis; left apical scar; general organized peritoneal adhesions; operative wounds.

The striking clinical features of this case are the absence of constipation, the indefiniteness of the bowel disturbance, the repeated presence of large quantities of indican in the urine, the unfavorable influence of a proteid diet, the persistent rapidity of the pulse in all of his attacks, the distress at the end of urination, the fact that perforation evidently followed distention of the sigmoid with water, the fact that a surgeon of high diagnostic ability operated for what seemed to be an acute attack of appendicitis, the interesting distribution of the tumor mass, which extended from the left iliac fossa down into the *cul-de-sac* and up into the right iliac fossa, where it reached its greatest prominence, and that this led a surgeon of large experience, after making a median exploratory incision, to drain the apex of the abscess in the right iliac fossa, with the belief that the seat of the trouble was there.

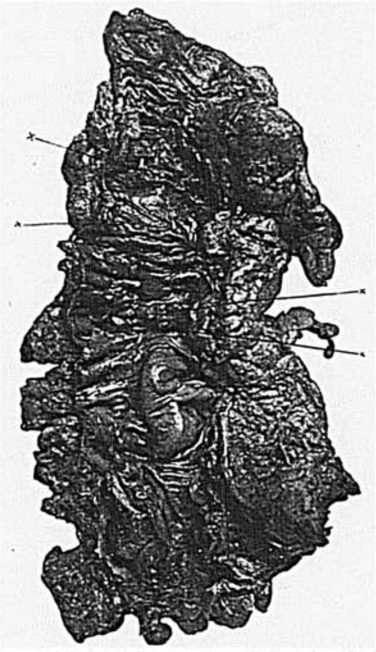


FIG. 4.—Photograph of the sigmoid of Case I, to show the site of the perforated diverticulum and the mouths of other small ones (X).

CASE II.—The following case was seen with Dr. Schleier, of Omaha. J. S., male, aged eighty-three years. The patient entered St. Joseph's Hospital December 9, 1908, in a semicomatose condition. No history could be obtained except that three days before admission he had had some trouble in passing urine and had, therefore, been catheterized. Subsequent urination had been free and the urine bloody.

General examination proved negative, except for a pulmonary emphysema and a well-developed arteriosclerosis. The abdomen was distended. Peristaltic waves could be seen starting in the region of the cecum, disappearing in the right hypochondrium, and again appearing at the median line, thence proceeding to the left and downward to the hypogastrium. There was no abdominal rigidity. There was tenderness posteriorly over both kidneys, with an area of renal hyperesthesia on both sides above the crest of the ilium. There was no abdominal tenderness. The bedsheets were stained with bloody urine; a specimen could not be obtained by catheterization. The breath was not urinous.

Rectal Examination. The rectum was ballooned, the prostate moderately enlarged, but not tender. No tumor mass was palpable.

Clinical Diagnosis. Chronic intestinal obstruction, probably due to stricture, cause unknown. Without a urine examination a chronic intestinal toxemia was suggested as a possible cause of the hematuria. Colotomy was advised and performed by Dr. Schleier. Death occurred about eighteen hours after operation.

Autopsy was performed eight hours after death. The patient was a somewhat emaciated elderly man. There was moderate edema and posterior congestion of both lungs, and marked senile emphysema. The heart was normal in size; there was tremendous calcification of the mitral valves and complete calcification of certain areas in the coronary arteries; there was also calcification of the aortic valves, and marked atheroma of the aorta throughout its entire length. The celiac axis was almost completely obliterated. The large intestine was dilated throughout its entire length; the hepatic flexure was adherent beneath and covered by the liver, a condition which explains why peristalsis could not be seen in this region. A colotomy wound was observed at lower part of descending colon. There was a stricture at the junction of the middle and lower third of the sigmoid. Numerous false diverticula were present in the area of the stricture, which was about one and one-half inches long (Fig. 5). There was a slight perisigmoiditis at the site of the stricture. Diverticula were numerous for a distance of a foot or two above the stricture. They varied in depth; their mouths were generally wide, and they entered the appendices epiploicæ. There was atrophy of the walls of the colon and of the small intestine. The kidneys were normal in size, the capsules stripped easily, and the surface was slightly granular. The cortex was atrophic, but there were no gross signs of inflammation. The pelvis and both kidneys were markedly injected and covered with a dirty, sanguinopurulent exudate. The ureters were distended and similarly inflamed. They were so red and injected as to look like veins from the outside. The bladder wall was thickened and markedly injected. The bladder contained a small amount of bloody urine which showed pus cells and desquamated epithelium in large amounts. There were microscopic clumps of a dozen or more epithelial cells together, showing that desquamation *en bloc* of areas of bladder, ureteral, and pelvic epithelium had occurred. The prostate was hypertrophied; there was marked peripheral arteriosclerosis, with extensive deposits of lime salts in the vessel walls. The pathological diagnosis was emphysema and passive congestion of the lungs; coronary arteriosclerosis and calcareous deposits in mitral and aortic valves; general arteriosclerosis; passive congestion of liver and spleen; senile atrophy of spleen; chronic intestinal obstruction

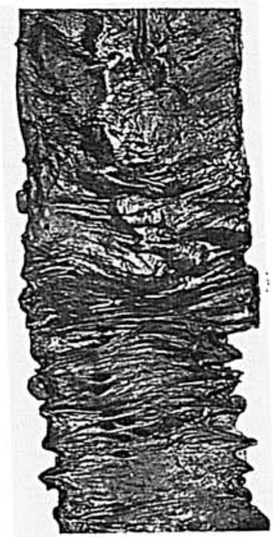


FIG. 5.—Photograph of the sigmoid of Case II, to show diverticula in the portion involved in the stricture.

due to benign cicatricial stenosis of sigmoid; false diverticula of the descending colon and sigmoid, especially at area of stricture; dilatation and atrophy of the colon; hypertrophy of the prostate; ascending cysto-uretero-pyelitis.

The intimate relation of the numerous false diverticula to the stenosed areas in this case makes probable their pathogenic significance in benign cicatricial stenosis of the sigmoid.

CASE III—S. B., farmer, aged fifty years, entered St. Joseph's Hospital January 4, 1909. The patient was in a stuporous condition, and the following history was obtained with difficulty. The duration of illness was indefinite and onset had been gradual. The patient complained of intermittent abdominal pain, which was described as "sore, colicky," of belching, headache, weakness, and of constipation. His bowels had not moved at all for three days, and not well for several weeks previously. No pulmonary, cardiac, or renal symptoms could be elicited. The past history was negative except for indefinite trouble with the lower bowel, and constipation. Later, a history was obtained of a disturbance, nine months previous, in the lower abdomen, which lasted several weeks, and was characterized by colicky pains and distress in the median line and to the left, alternating diarrhea and constipation with mucus in the stools, fever, and soreness in the left lower quadrant. The condition of the patient when first seen was as follows. He was an emaciated, medium-sized man, stupid in appearance, and apparently of some sixty years of age. He lay quietly in bed, yawned continuously, his eyes were half closed, and he paid but little attention to what was going on around him. It was necessary to speak loudly and prod him to obtain answers to questions. Both lids were equally ptotic, but he could raise them with the assistance of the frontal muscles to the level of the iris. The pupils were equally contracted, and reacted slightly to light and distance. The breath was foul, and the teeth poor. Vision seemed good. There was no asymmetry in muscle power. The patient could recognize a watch tick for a distance of only about an inch from each ear. Examination of the neck, lungs, and heart was negative. The abdomen was soft and not especially tympanitic. The liver and spleen were negative. Peristaltic waves occasionally could be seen traversing the hypogastric region from right to left. They were about two to three inches in breadth and were accompanied by signs of distress and rubbing of the abdomen. Rectal examination, save for "ballooning" was negative. No tumor mass was palpable. The urine was highly concentrated. It contained indican and urobilinogen (Ehrlich's aldehyde reaction) in large amounts, a trace of albumin with occasional hyalin, and granular casts. The superficial reflexes were markedly diminished, and the deep reflexes were faint or absent, but symmetrically so. The temperature was 101°, and the pulse 70. An injection of warm olive oil was ordered, with a mercurial and saline, which brought away a large amount of fecal material. The mental condition brightened for several days. Then relapses occurred at varying intervals with conditions similar to those described. The trouble always could be relieved by a rectal injection of one to two quarts of water. A diagnosis of chronic intestinal obstruction, of unknown cause, in the region of the sigmoid, was made at this time. On January 25, 1909, the patient developed a double lobar pneumonia, which lasted ten days. During this time injections of water were given each day.

On February 28, 1909, another attack of pneumonia occurred which produced stupor and was accompanied by signs of intestinal obstruction, *e. g.*, peristalsis and obstipation, which were relieved only by repeated injections of both oil and water. A large sacral decubitus four inches in diameter developed at this time. Several attacks of obstruction followed, which were easily remedied by injections. During intervals of from one week to ten days the patient had normal although constipated stools.

It was clear that an intermittent intestinal obstruction existed. This must have been caused by a mechanical factor prevailing only at intervals. The occasional copious and well-formed movements spoke against stricture. The constant absence of blood in the stools, and of a tumor mass, spoke against malignant disease. There was no evidence of hernia. Of causes that could produce such a frequent recurrence of obstruction, followed by free intervals, only two could well be assumed. The first was adhesions. But a band could hardly produce this picture by mere constriction. Traction of an adhesion on a loop of intestine might cause kinking when influenced by changes in intra-intestinal pressure, and by variations in the position and caliber of the gut, both of which factors are, to a certain extent, dependent on the intestinal contents. The second possible cause was intermittent volvulus upon the basis of cicatrization and retraction of the mesosigmoid forming an axis on which rotation of the sigmoid might occur. Such a condition might have accounted for an intermittent intestinal obstruction, which was not relieved by catharsis, but by rectal injections. A history of intermittent intestinal trouble with fever, pain, and diarrhea, with the presence of blood the year before, suggested the possibility of such a condition.

Operation was advised on the basis of a diagnosis of intermittent chronic intestinal obstruction, due to volvulus or to adhesions. An old inflammatory process resulting from a diverticulitis was suggested as a possible *causa morbi*.

The operation was performed by Dr. C. C. Allison. An incision was made to the left of the median line. The upper and lower segments of the sigmoid were found separated by about one inch of scarred mesosigmoid. There were slight adhesions running from the origin of the sigmoid to the parietal peritoneum. Several diverticula were seen close to the mesenteric attachment in the vicinity of the retracted mesosigmoid. It could not be demonstrated without opening the gut, that the cicatrization was secondary to a diverticulitis, and so, although the existence of the diverticula may have been a coincidence, yet inference to the contrary is permissible. The upper limb of the sigmoid was freed from adhesions and stitched to the parietal peritoneum higher up. A complete recovery took place.

CASE IV.—N. A. C., male, aged seventy-five years. The case was a coroner's autopsy. The anatomical diagnosis was: Myomalacia cordis; sclerosis and obliteration of the right coronary artery; general arteriosclerosis; passive congestion of liver, spleen, and kidneys; chronic interstitial nephritis; pulmonary emphysema. Several false diverticula of the sigmoid were found entering the appendices epiploicæ.

CASE V.—E. R., male, the anatomical diagnosis was: Healed tuberculosis of both apices; chronic bilateral pleural adhesions; dilatation and hypertrophy of the heart; myomalacia cordis with cardiac aneurysm; passive congestion of lungs, liver, kidney, and spleen; cholelithiasis; arteriosclerotic kidney; diverticula of the sigmoid and descending colon. The diverticula were located in the vicinity of the mesenteric attachment. Their walls were extremely thin, and they contained inspissated feces (Figs. 6 and 7).

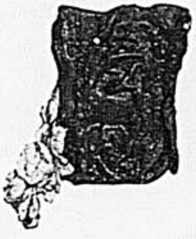


FIG. 6

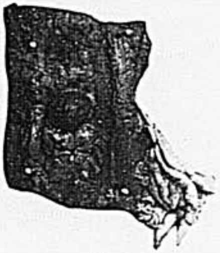


FIG. 7

FIG. 6.—Photograph of one of the diverticula in the sigmoid of Case V.

FIG. 7.—Photograph of the same diverticulum in Case V, showing the inspissated feces contained in it.

CASE VI.—The case was a coroner's autopsy. The subject was an elderly obese male, of unknown age. The anatomical diagnosis was: Edema of lower extremities; passive congestion and edema of the lungs; hypertrophy and dilatation of the heart, especially of the left ventricle; degeneration of the myocardium; passive congestion of the liver, spleen, and kidneys; chronic interstitial nephritis; false diverticula of the sigmoid. The sigmoid was surrounded with a large amount of fat, and the appendices epiploicæ consisted of large fatty masses. Six false diverticula could be seen penetrating these fatty masses (Fig. 8).

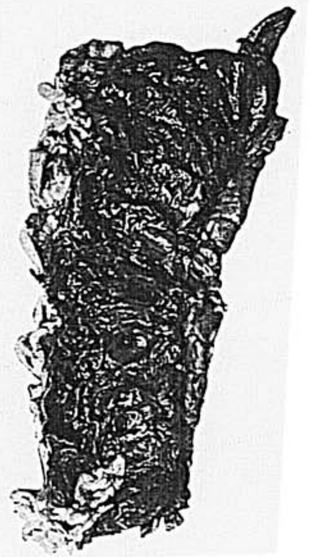


Fig. 8.—Photograph of a portion of the sigmoid of Case VI, to show diverticula.

Diagnosis. It can be easily seen, from the brief *resume* of the pathology of diverticulum disease, that the symptoms may be manifold and difficult of interpretation. The diagnosis is seldom easy, rarely certain, and often impossible. Franke was able to collect from the literature only 2 cases in which the diagnosis had been made. To these he added a case of his own. Graham recently suggested the diagnosis in one of Mayo's cases. To appreciate the possibilities is a long step in the direction of a diagnosis, which must be made largely by exclusion. The most easily recognizable condition is that of the so-called "left-sided appendicitis." There is pain, gastric disturbances, tenderness, and rigidity in the left lower quadrant, with temperature and leukocytosis; abscess formation often appears later. Another class of cases that is moderately easy of recognition is that of chronic hyperplastic sigmoiditis. This has been treated as a disease entity by Ewald, Boaz, Rosenheim, and others, but it is possible that diverticulitis may be found a basis for it. In this complex there is always intermittent pain, disturbed function associated with irregularity of bowel action, gas, and often spastic stools in which there is frequently an increase in mucus and occasionally a trace of blood. Physical examination discloses a tender sausage-shaped tumor which may be either adherent or freely movable, and which varies in size from time to time. Infiltration may be felt occasionally per rectum. Sigmoidoscopy shows a swollen, reddened, rugous mucosa, and often narrowing of the lumen of the gut. Obstructive symptoms may be present, but are not so common as in cancer, from which condition it must be differentiated. In carcinoma the clinical course is more progressive and definite. Its duration is limited; ulceration with blood in the feces is the rule, and when blood once appears it is usually quite constant. The tumor is harder, more likely to be movable, definitely circumscribed, constantly increases in size, and is not elongated. Bladder symptoms are apt to be frequent in diverticulitis, as the bladder is often involved in the inflammatory process. Cripps states that vesicosigmoidal fistula is more commonly inflammatory than malignant (45 out of 63 cases). If such be the case, many of them may be the result of a diverticulitis. Disturbed bowel function is constant. Many cases record constipation as a more or less pronounced prodromal symptom. Pelvic abscess, tuboövarian abscess, mucous colitis, amebic colitis, chronic proctitis, tuberculosis and syphilis of the sigmoid, psoas disease, retroperitoneal abscess in the left flank from other causes, etc., must, in addition to the conditions already mentioned, be differentiated.

TREATMENT.—The treatment must be surgical in many cases. The prognosis is often a question of technical efficiency. It is certain that, as in appendicitis, many of the cases will recover without operative interference. Just what are the indications for resection in hyperplastic sigmoiditis is a question for future determination. Abscesses and vesicosigmoidal fistulæ should always have the advantage of surgical treatment. As knowledge of the morbid anatomy of false diverticula increases, as cases are recognized, reported, and treated, as methods of surgical procedure become crystallized, facts will be obtained for reliable diagnosis and prognosis.

LITERATURE.

Fischer. Jour. Exper. Med., 1900, v, 33.
Beer. Amer. Jour. Med. Sci., 1904, cxxviii, 135.
Telling. Brit. Med. Jour., October 31, 1908, 2196.
Franke. Deut. med. Woch., 1909, xxxv, 98.
Eisendrath. Arch. Diag., October, 1909.
Giffin and Wilson. Amer. Jour. Med. Sci., 1909, cxxxviii, 661.